DELAWARE STATE MEDICAL JOURNAL

Official Organ of the Medical Society of Delaware
INCORPORATED 1789

VOLUME 24
NUMBER 6

JUNE, 1952

Per Year, $4.00
Per Copy 50c

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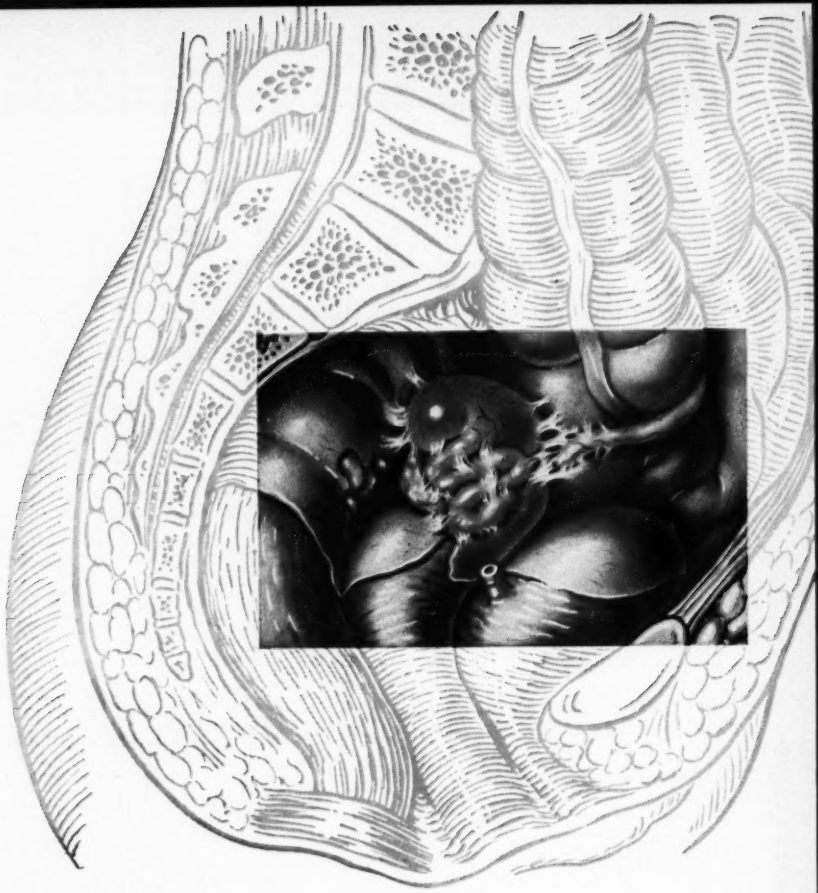
Entered as second-class matter June 28, 1929, at the Post Office at Wilmington, Delaware, under the Act of March 3, 1879. Editorial Office, 822 North American Building, Wilmington 7, Delaware. Business Office, Farnhurst, Delaware. Issued monthly.



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1. Greene, G. G.: Kentucky M. J. *50*:8, 1952.
2. Stevenson, C. S., et al.: Am. J. Obst. & Gynec. *61*:498, 1951.

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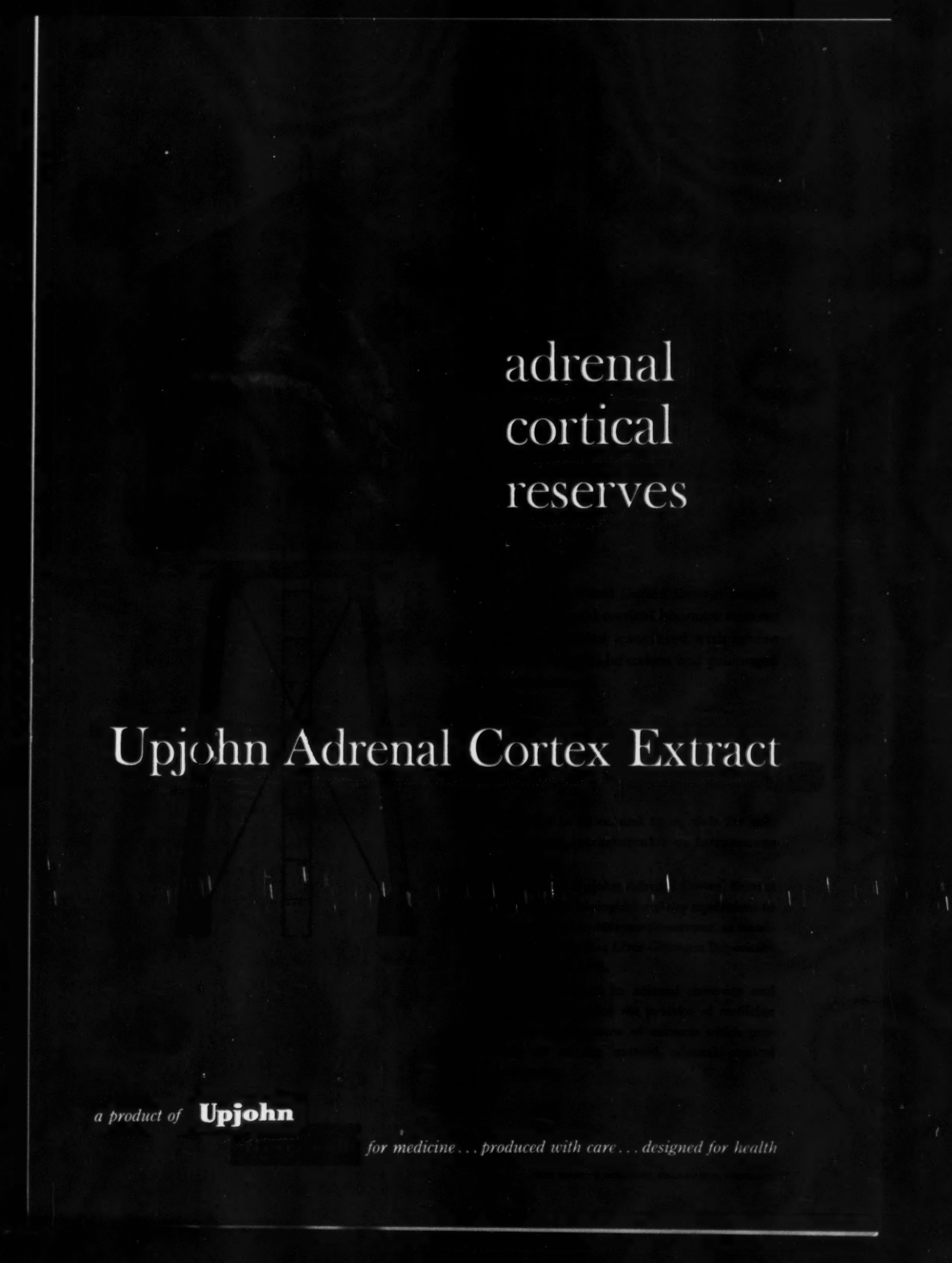
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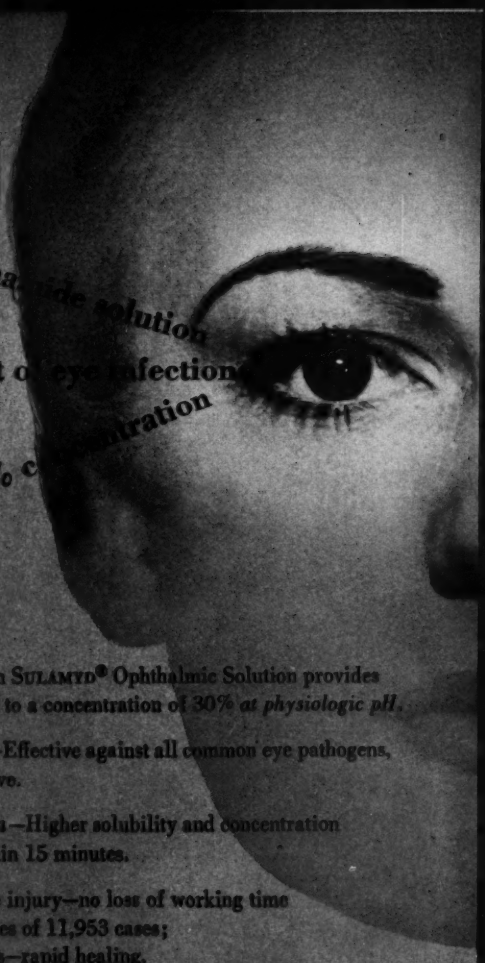

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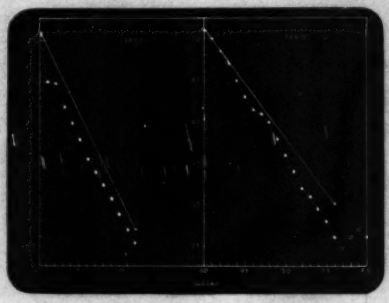

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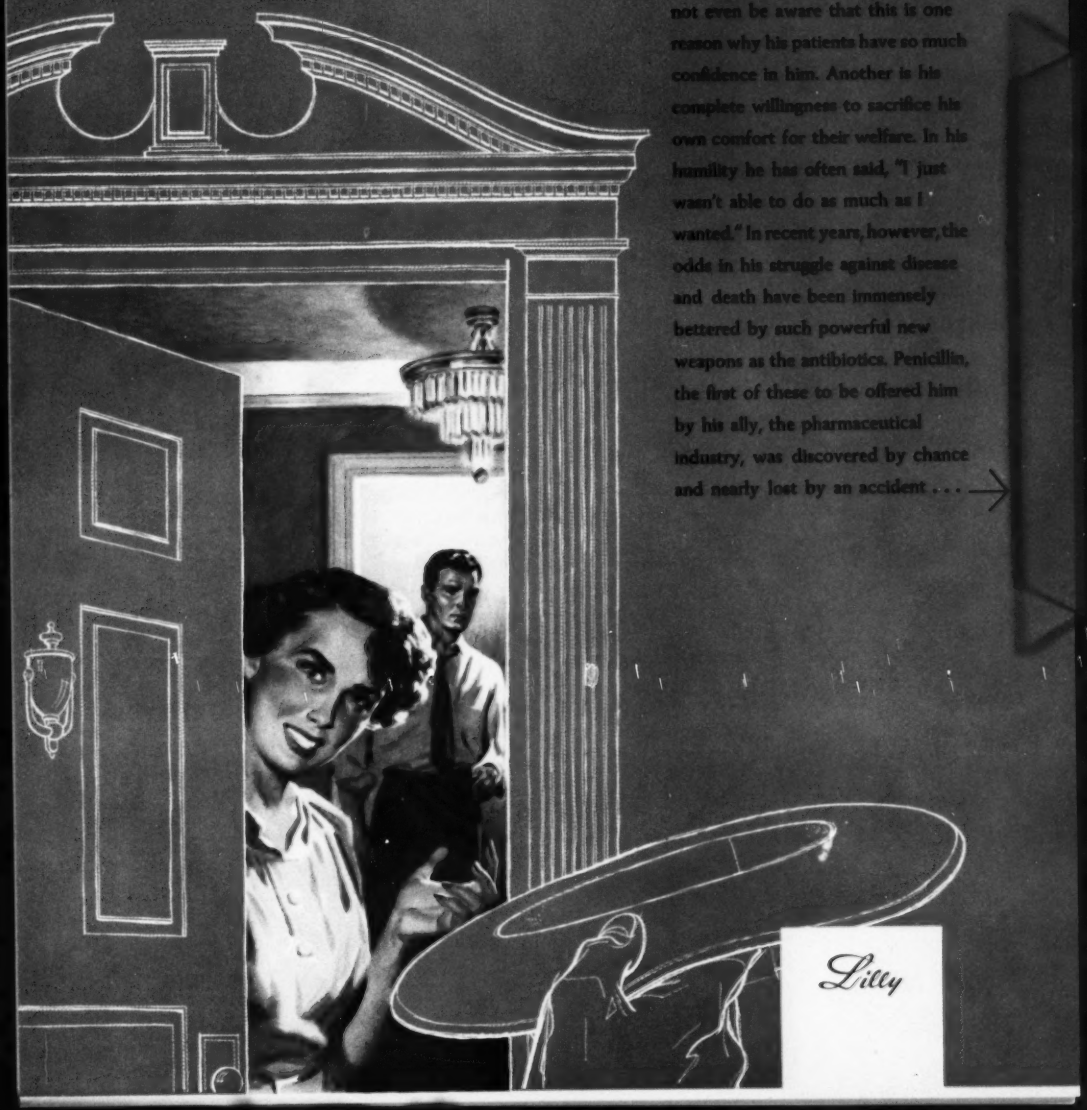

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DELAWARE STATE MEDICAL JOURNAL

Issued Monthly Under the Supervision of the Publication Committee
Owned and Published by the Medical Society of Delaware

VOLUME 24
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CRANIOCEREBRAL TRAUMA*

RICHARD G. COBLENTZ, M. D.,**
Baltimore, Md.

It is an established fact that, in the past decade, trauma to the head has become one of the major causes of death. R. Glenn Spurling has stated: "The number of severe head injuries has increased in direct ratio with each increase in the speed of transportation." It is true also that the number of qualified specialists in neurology and neurosurgery has increased greatly in the past decade. Even so, because of the wide distribution of severe head injuries, it is obvious that only a small percentage of them can be cared for by these qualified specialists. Every general practitioner and every general surgeon in every locality will at one time or another be called upon to treat a patient with craniocerebral trauma. The manner in which the patient is treated is all important and while frequently determining survival or decease of the patient, always determines his future value to society.

It is imperative, therefore, that the general practitioner and especially the general surgeon, be familiar with the broad general principles upon which the modern treatment of head trauma is based. These principles are based on a knowledge of the basic anatomy, physiology and pathology of the brain. A short review of the pertinent anatomic, physiologic and pathologic data will therefore be considered before proceeding with classification, diagnosis and treatment.

From an anatomical standpoint, the skull, the membranes covering the brain and spinal cord. The great venous sinuses lying between folds of the dura, the middle meningeal artery and the cerebrospinal fluid are most important.

The skull is a rigid container which cannot expand and contract to accommodate for changes in volume of its contents. The foramen magnum at the base of the skull is a large opening through which the brain stem joins the spinal cord. Many important medullary structures are located in this area. Both of these facts are important from the standpoint of both generalized and focal pressure on neighboring structures. At the same time the skull is so constructed that it can transmit force to the cranial contents without being fractured. For this reason fatal brain injuries can be produced without demonstrable skull fractures.

The membranes covering the brain are the dura mater, the arachnoid and the pia mater. The dura mater is a tough fibrous structure made up of two layers. The outer layer forms the endosteal lining of the cranial bones and the inner covers the brain. One extension of the dura forms a longitudinal partition between the two cerebral hemispheres. A second extension forms a horizontal partition separating the cerebellum from the cerebral hemispheres. An opening in this partition is in the region of important structures. Large venous sinuses lie between folds of the dura. The superior longitudinal and the two lateral sinuses receive venous blood from the brain through thin walled cortical veins. The sinuses empty into the internal jugular veins. Trauma in the region of these large sinuses may cause rupture of the sinus itself or of the veins emptying into it, and thereby produce a collection of blood in the subdural space to form a subdural hematoma.

It is extremely important that one be familiar with the course of the middle meningeal artery, which is frequently involved in head trauma. It enters the base of the skull through the foramen spinosum and runs through a bony groove in the temporal bone from which it is distributed to the dura mater. This artery is frequently torn by linear fractures in the temporal bone. Arterial bleeding thus produced accumulates between

* Read before the Medical Society of Delaware, Wilmington, October 9, 1951.

** Clinical Professor of Neurological Surgery, University of Maryland.

the dura and the skull. A clot thus formed dissects the dura from the skull and gradually enlarges to form an extradural hematoma, one of the most important surgical complications of head trauma.

The cerebrospinal fluid is secreted chiefly from the chorioid plexus in the lateral ventricles. It passes through the ventricular system to the cisterns at the base of the brain. It then flows over the surface of the brain in the subarachnoid spaces. Most of the fluid is then absorbed into the venous sinuses through the arachnoidal villi and granulations. Normal absorption may be altered by blood in the spinal fluid circulating in the subarachnoid space. The cerebrospinal fluid acts as a space compensating mechanism between the brain and the skull. Blood in the spinal fluid acts as an irritant and produces signs and symptoms characteristic of subarachnoid hemorrhage.

One should be familiar with the physiology of the cerebral cortex in order to correctly interpret neurologic signs which may be produced by injuries to the brain itself. Some of the important gross areas of localization include the frontal poles, the motor and sensory areas, the speech center, the visual centers and the basilar structures of the brain. Damage to the frontal poles causes alteration of cerebration, concentration and judgment. Lesions of the motor cortex produce variations of voluntary muscular activity. Generally speaking destructive lesions cause contralateral hemiplegia while irritative lesions of cortical cells produce Jacksonian epileptiform seizures on the opposite side of the body. The sensory area lies directly behind the motor area. Damage in this area causes sensory disturbances on the opposite side of the body. The speech area is localized in a wide area centered about the junction of the Rolandic and Sylvian fissures in the left hemisphere in the right handed and the right hemisphere in the left handed. The anterior portion is chiefly motor. Extensive lesions in the speech area produce both motor and sensory aphasia. The visual centers are located in the occipital poles in the Calcarian cortex on the mesial surface. Damage in this area produces visual field defects depending on the size of area involved. Damage to the basilar structures in the region of the hypothalmus and mesencephalon can produce alterations in blood pressure, water regulation and temperature—producing glycosuria, hyperemia and death.

Numerous classifications of head injuries have been made, some based on the principal of clinical entities, others on the physiological or anatomical conditions present. The following classification is based on the resultant gross pathology and is an abstraction from personal experiences and those in the literature.

1. Simple scalp wounds:
   Brush burns, contusions, lacerations, subgalea hematoma.
2. Skull fracture (with or without associated brain injury)
   - A. Vault
   - B. Base
   - C. Simple (linear or comminuted)
   - D. Depressed
   - E. Compound
3. Brain Injury (without or without associated skull injury)
   - A. Concussion
   - B. Oedema (cerebral)
   - C. Cerebral contusion
   - D. Cerebral laceration
   - E. Hemorrhage
     - (1) Extradural
     - (2) Subdural
     - (3) Intracerebral
     - (4) Subarachnoid
     - (5) Diffuse petechial
4. Combinations of the above.

Subsequent discussion largely rests on this classification.

No routine procedures can be followed in the initial stage of a serious head injury. The initial symptomatology will vary in almost every case. Often the later course of individuals with exactly similar initial symptoms will vary. Every means should be exhausted to ascertain the condition of the patient immediately following the injury and in the interval which may have elapsed before seen by a physician. If this interval is a matter of hours the information obtained may be the only guide for the proper diagnosis and treatment. The manner in which an injury is sustained is also important from a diagnostic standpoint.

Symptoms of traumatic shock are infre-

quent in uncomplicated cases of head trauma. If shock is present one should examine carefully for other injuries such as fractured bones, including cervical spine, chest injuries, ruptured viscus with intrathoracic or intraperitoneal hemorrhage and chest injuries. When signs of shock are observed, the treatment of shock takes precedence over every other consideration.

Such matters having been taken care of an effort should be made to classify the injury and to determine as soon as possible what type of operative or non-operative treatment is indicated.

Generally speaking there are certain injuries which fall into the non-operative group. Namely:

1. Simple concussion
2. Cerebral oedema
3. Cerebral contusions and lacerations uncomplicated by extradural, subdural or intracerebral hemorrhage producing increased intereranial pressure.
4. Uncomplicated subarachnoid hemorrhage
5. Simple linear fractures of base of the skull without associated brain injury.

Those requiring operative treatment include:

1. Scalp lacerations
2. Depressed fractures of skull, both simple and compound
3. A few linear fractures complicated by dural tears producing cerebrospinal fluid rhinorrhea
4. Extradural hematoma
5. Subdural hematoma
6. Penetrating wounds of brain
7. Intracerebral clots (encysted)

Careful observation and repeated neurological examinations together with certain indicated diagnostic procedures such as spinal puncture, x-ray examination, electroencephalographic studies and occasional air studies are used to make a differential diagnosis. Important neurological signs include

1. The depth and duration of unconsciousness
2. The presence of paralysis
3. The presence of decerebrate rigidity
4. Convulsions
5. Cervical rigidity
6. Alterations in pulse, respiration, temperature and blood pressure
7. Occular signs
8. Vertigo
9. Mental symptoms

By and large, the degree of cerebral trauma is in direct ratio to the depth and length of unconsciousness. The length may vary from a few minutes to several weeks. Concussion is clinically characterized by a transient impairment or loss of consciousness which may last from a few seconds to several hours. Having regained consciousness, most usually having vomited, the patient makes a speedy and complete recovery. The spinal fluid is clear under normal or increased pressure. A patient who has not regained consciousness after two or three hours has probably sustained contusions and lacerations of the brain.

A contused brain implies a bruised brain with moderate bleeding into the subarachnoid space with normal or increased spinal fluid pressure. Cerebral laceration implies a torn brain with varying amounts of bleeding into the subarachnoid or subdural space. Without gross bleeding the spinal fluid pressure is normal or lower than normal. The most frequent points of laceration and contusion are at the tips of the frontal, temporal and occipital poles. The opposite side of the brain to the point of impact may be contused or lacerated by contra coupe.

Cerebral oedema is due to an increase of tissue fluids in the brain. Recent experimental studies indicate that this condition is not a frequent condition following trauma. When it does occur, uncomplicated, the spinal fluid is clear and under increased pressure.

Subarachnoid hemorrhage, which implies bleeding into the subarachnoid space, is usually followed by irritative signs such as restlessness, delirium, neurologic like head pains, cervical rigidity and all the classical signs of meningitis. The spinal fluid contains blood and the pressure is normal or less than normal when gross hemorrhage is present.

Patients with small deep seated multiple hemorrhages show characteristic signs of low spinal fluid pressure: rapid pulse and respiratory rate; sharp rise in temperature; semiconsciousness with restlessness; spasticity of muscles, cranial nerve or other paralyses; lethargic state and other neurologic signs, depending on the location of the lesions—the most frequent sites being in the region of the

Vein of Galen system, the brain stem and the basal ganglia.

The important measures to be considered in the non-operative treatment include:

1. The control of shock
2. The maintenance of free respiratory exchange and the prevention of drainage of mucous, vomitus and secretions into the bronchial tree. This is best accomplished by postural drainage. The foot of the bed is elevated to an angle of 30°. The patient is placed on the abdomen or side, with the head in such position to permit free drainage.
3. The control of restlessness by sedative drugs and limited restraint. The drugs which have been found to be most effective include paraldehyde by mouth or rectum; sodium luminal in fairly large doses hypodermically or sodium amytal intravenously. Morphia is contraindicated because of its depressing effect on the respiratory centers and its masking effect on the state of consciousness. Often the catheterization of a full bladder will produce remarkable results in controlling restlessness.
4. Nourishment in the unconscious patient should be maintained by intravenous injections of dextrose 10% solution in sterile water or by a high caloric, well-balanced intake by gavage.
5. The question of fluid intake is still a most disputed question. Some advise extreme dehydration. Others feel that this is unnecessary in most patients and that it may produce confusing elevation of temperature, and an alarming high protein nitrogen. We have been convinced that best results can be obtained by limiting the fluid intake to 1500 cc per 24 hours. 10% dextrose in distilled water intravenously is recommended.
6. The question of spinal puncture is also a disputed one. In some clinics spinal puncture is performed on every case of craniocerebral injury regardless of the clinical picture. Others are more conservative and recognize certain contraindications. Numerous proven fatalities have been reported following spinal tap on patients with increased intercranial pressure. Pressure cones may be produced at the incisura or the foramen magnum causing death or alarming medullary symptoms. We feel that lumbar puncture is indicated in cases which do not show clinical evidence of increased intercranial pressure, mainly to determine the presence or absence of blood. At times repeated punctures are indicated for the removal of bloody spinal fluid where the bleeding has ceased and the blood is causing meningeal irritation. Spinal puncture is contraindicated in all cases where there is clinical evidence of high increased spinal fluid pressure or of expanding lesions in the posterior fossa. Bilateral exploratory trephines under local anaesthesia is a simple and safe procedure and should be used for diagnosis and localization in such cases.
7. The use of hypertonic solutions to reduce intercranial pressure is reserved for those patients who are in critical condition from surgical complications and when time is needed to prepare the patient and the operating room for surgery, or for those patients who develop increased intercranial pressure from meningeal irritation without surgical complications. Hypertonic sale solution or concentrated dextrose will reduce the pressure quickly. For prolonged use concentrated sucrose (50%) is effective but is not without danger of renal damage.

One of the most important operative procedures in craniocerebral trauma is the proper treatment of scalp wounds. All scalp wounds should be handled meticulously for even the small puncture wounds act as portals for infection. The important steps to be followed are:

1. Careful cleansing and shaving of a wide area about the wound, and the wound itself after blocking off with local anaesthesia.
2. Adequate draping
3. Inspection and palpation of pericranium and skull for fractures
4. Control of hemorrhage
5. Careful debridement
6. Accurate primary suturing of the wound in layers, using interrupted black silk sutures in the galea and through and through interrupted sutures in the scalp
7. The use of sulphonamides and antibiotics to prevent infection.

It should be kept in mind that the improper care of a simple scalp wound may result in serious complications such as osteomyelitis—cerebral abscess or meningitis.

Practically all depressed fractures of the skull, both simple and compound, require surgical repair. This type of injury may be associated with dura tears and local brain contusion and laceration which will also require repair. The principles to be followed include those mentioned for scalp wounds and in addition careful removal of detached bone fragments and foreign material, adequate exposure for inspection and repair of the underlying structures and the control of hemorrhage, removal of devitalized brain tissue by suction and careful closure of the dura to prevent spinal fluid leak. Large bony defects should be left for a secondary operation. Some small defects can be repaired at the time of the initial operation, depending on the condition of the patient and the facilities at hand.

Extradural hemorrhage almost always has its origin from a tear in the middle meningeal artery usually produced by a linear fracture extending across the squamous portion of the temporal bone. The bleeding is usually slow as it strips the dura from the skull, but it may expand rapidly to cover a large area of the hemisphere to produce increased intercranial pressure and focal neurological signs. The classical syndrome may manifest itself by a short period of unconsciousness following a blow on the head, followed by a lucid interval in which the patient may complain of headache and nausea but be perfectly rational. This interval in turn is followed by other signs of increasing intercranial pressure: drowsiness, stupor, coma, slowing of the pulse, vomiting and elevation of blood pressure. This state may be preceded or followed by focal signs such as weakness of the contralateral face, arm and leg with reflex changes and a dilated pupil usually on the side of the lesion. If the condition is not recognized and treated the coma deepens—there may be a convulsive seizure and the pulse and temperature increase and the blood pressure falls. Medullary function fails and the patient dies within several hours. Long before these symptoms develop a diagnosis should be made and operation performed for removal of the clot and the control of hemorrhage. Doubtful cases should be explored early. The original burr opening on the suspected side at the temporal ridge will confirm the diagnosis and serve as a starting point for a liberal subtemporal exposure for the removal of the clot and the control of bleeding.

Subdural bleeding usually arises from a tear of one or more of the cortical veins entering the venous sinuses. In severe head trauma, laceration and contusion of the brain may be present and therefore bleeding from the cortical vessels may be either venous or arterial and difficult to control. Bleeding spreads unrestricted because of the nature of the subdural space. It may consist of a thin soft clot over a portion of the cortex or it may be found as a large collection of blood over one or both hemispheres. In most cases the arachnoid is torn and the leakage of cerebrospinal fluid produces a mixture of blood and spinal fluid. This accounts for the fact that solid subdural clots are not often encountered. In the acute form the symptoms vary widely and depend on the location of the brain injury and the extent of bleeding. Most patients are unconscious after the injury and remain so. Signs of increased intercranial pressure develop with or without focal signs, depending on the location of the lesions. When one is forced to treat the patient surgically at an early stage, the treatment is often unsatisfactory due to the fact that the venous or arterial bleeding is difficult to localize and control.

Often multiple exploratory burr openings are necessary to locate and evacuate the blood. Drainage is frequently necessary. Improvement is usually slow after operation. The control of increased intercranial pressure and the use of non-operative treatment measures are the main points to be considered. Re-exploration for fresh bleeding is frequently necessary when there is a secondary rise in intercranial pressure.

Chonic subdural hematoma presents a different picture clinically. This condition usually follows a slight trauma to the head which produces a small tear in a cortical vein entering the venous sinuses without other brain damage. The bleeding is slow and intermittent allowing the brain to accommodate

itself to a gradual increase in intercranial pressure without definite early signs. Symptoms may appear within a few days but usually weeks or even months elapse before symptoms appear and the condition is suspected. During this period the blood becomes encapsulated and may extend over the entire hemisphere or may be localized over a smaller area.

The symptoms consist of personality changes, mild intermittent headache, drowsiness and mental retardation. Later, as the intercranial pressure slowly increases, nausea, vomiting, dizziness and blurred vision develop. Convulsions and localized paralyses are not frequently encountered.

The neurological signs are those of slowly increasing pressure. Focal signs are variable and slight and therefore cannot be relied upon for diagnosis or localization. The spinal fluid pressure is slightly elevated and slightly xanthochromic.

Exploratory trephine openings will establish the diagnosis when the exposed dura presents the typical greenish-blue appearance. When the outer wall of the sac lying on the under surface of the dura is opened, brownish-red liquid clots, often in large quantities, evacuate themselves. The trephine opening should be slightly enlarged to permit adequate opening in the dura and sac to permit free evacuation to effect a cure without drainage. Occasionally solid clots and clots in all stages of degeneration are encountered. In such cases when the clot cannot be removed it is necessary to turn a bone flap and remove the entire sac with its contents. It is not necessary to remove the sac to effect a cure. Bilateral exploration is always indicated, for the condition is bilateral in 20% of the cases.

The treatment of penetrating wounds of the brain is based on the principles developed by Dr. Cushing and his co-workers in World War I. Only a few modifications and refinements have been added. These principles briefly consist of

1. Thorough cleansing of the area surrounding the wound as mentioned in treatment of scalp lacerations
2. Enlargement of the scalp wound to expose the bony involvement
3. Enlargement of bony involvement to give adequate exposure of involved brain
4. Careful debridement of all structures from without inward
5. Control of hemorrhage by coagulation or silver clips
6. Removal of all macerated brain, small bone fragments and debris by suction and irrigation
7. Removal of metallic bodies when feasible and possible without large damage to the brain
8. Repairs of dura with fascia or prepared membrane to effect a tight closure
9. Closure of scalp without drainage as described in scalp wounds
10. Use of chemotherapy and antibiotic therapy to prevent infection

The operative treatment of cerebrospinal fluid rhinorrhea is a formidable procedure and should be left to the trained neurosurgeon and avoided by the occasional intercranial operator.

Time does not permit a discussion of the common sequela, of craniocerebral trauma. It is hoped that such sequela, as epilepsy, cortical atrophy and the so-called post concussion syndrome will be less frequently encountered as the general principles of treatment are more frequently employed.

In this paper I have made no pretense of thoroughly covering all aspects of craniocerebral trauma. Neither has an attempt been made to discuss in detail the non-operative treatment or operative technique. It has been written primarily to stimulate more detailed investigation by the physician and surgeon who is not himself a neurologist or neurosurgeon.

2 E. Read Street

DISCUSSION

DR. P. J. GORDY (Wilmington): I would like to thank Dr. Coblentz for this clear and interesting paper on the general care and approach to head injuries. He has covered the entire field.

I would only like to perhaps re-emphasize several points that he has mentioned. One is the extreme importance of the proper care of scalp wounds. As he says, it is too often forgotten that a scalp laceration is a portal of infection which may lead to serious complications, such as osteomyelitis, cerebral abscess, or meningitis.

In the approach to fluid balance in the

head-injured patient, I think the general trend, as Dr. Coblentz has pointed out, is toward leaving behind the older methods of extreme dehydration. Extreme dehydration in the patient who has been restricted in his fluid intake can be a striking thing and give a picture almost as alarming as was the head injury itself. So only an adequate fluid intake should be given.

Probably the most important single thing in the non-operative care of the head injury is maintenance of a clear airway, going as far as reinserting trachea tubes in an attempt to clear the bronchial tree.

In diagnosing time for surgical intervention in head injuries, the list or group of factors, as Dr. Coblentz has mentioned, should be, of course, interpreted as a whole. No single sign gives us the "pitch" on when to go in. I think the most, or among the most important of those, is the depth of unconsciousness of the patient, which depends on the careful observation from the time he enters the hospital so that you can decide whether or not the state of consciousness is deepening, staying the same, or improving.

The question of subdural hematoma of course, divides itself into the acute and chronic subdurals.

One thing might be mentioned in addition to Dr. Coblentz' discussion—that is, the presence of subdural hematomas in infants, bringing up a different problem but a very important one, in diagnosing these adequately and sufficiently early, so that the fluid can be removed—and, when necessary, the membranes are removed in order to prevent interference to the devolping of the infant's brain.

Chronic subdural hematoma has, as Dr. Coblentz mentioned, a minimum of definite signs. It might be called "the great imitator".

With regards to technique, I would like to say only this one thing. There is no harm in performing a negative trephine. This can be done under local or at the most under slight anasthesia, and it is better to perform a trephine rather than let a possible lesion go by. Many times it is necessary to do that even though we can make a clear preoperative diagnosis of a cranial lesion. It is necessary to say when the trephines should be bilateral, and one may not suffice. You may have to make several.

As an illustration, while in the Army at Frankfurt, we had a boy come in who died shortly after admission. He had been trephined at an earlier admission but, unfortunately, the trephining had not been sufficient to uncover the source of the lesion. Had another trephine been made, say, only 3 cm.'s from the orignial one, there may have been a different outcome.

DR. D. J. PRESTON (Wilmington): I am highly grateful to Dr. Coblentz for his excellent and practical discussion of an important subject. It brings to mind an early experience of mine in the treatment of head injuries.

I was asked to see a fifty year old man who had fallen down and injured his head. He was temporarily unconscious, apparently well for two days or so and then would become unconscious again. He was brought into the hospital in a comatose state. When I saw him he had signs of localization in his left hemisphere, with paralysis of the opposite side. That seemed like an obvious enough history. We took him to the operating room, made a hole, removed the blood clot and while on the operating table his pulse rate improved, and his blood pressure and respiratory rate improved. After he was returned to bed, he seemed to do well for the first day; then, despite supportive measures within two more days he died. I was very much disappointed, especially so because I was unable to obtain a post-mortem examination.

Soon after this occurred, I had occasion to discuss the case with a neurosurgical friend of mine and I said: "why was I unable to save this man?" He chuckled and remarked, "You probably would have saved him if you had opened the other side."

His feeling, of course, coincided with what was said today, and is a practical point that one trephine may not be adequate, and in all cases, if you don't find the clot that you know is there, you must certainly try elsewhere, there, or on the opposite side, or in different places on the same side.

The remarks made by Dr. Coblentz about the magnitude of scalp wounds are very good. I think any carelessness in this matter is inexcusable, because of the underlying chance

of cranial infection. It gives the examiner a chance to examine the underlying bone to determine presence of a fracture or indriven bone fragment. There I think we should take advantage of the debridement of the bone, and it is important that an aseptic technique be the rule, without question.

The penetrating wounds of the skull, especially as in shell fracture, etc., we don't see often in civilian life, but when they do occur the first thing the family will ask is: "has the bullet been removed?"

I was favorably impressed by Dr. Coblentz' remarks that it is not of great importance to remove the body that is not causing damage, and I am certain others, as well as he, have seen damage occur from unwise removal of metallic bodies not causing damage. The removal of such bodies can sometimes cause severe damage. This does not mean that intracranial wounds should not be debrided, with the removal of the chronic tissue and ingrown bone fragments, or in-driven fragments. I recall one doctor said "a bullet which enters the skull is dangerous only so long as it is moving." Perhaps that is not entirely true, but certainly the bullet if not moving and not causing damage might better be left alone.

The habit of rushing patients from the scene of the accident right to the x-ray room is sometimes unwise, especially in those who have severe injuries other than their head injury. I have seen, on occasion, a patient in shock, being taken to the x-ray department for x-ray studies and the treatment of the shock being delayed or deferred. That, I think is something we should guard against because certainly our first concern should be the treatment of shock. It is also true that in the case of many head injuries shock is relatively slight or absent, and when we see a patient with a deep shock and having a head injury, we immediately look for other causes for that shock—a crushed chest, intrathoracic bleeding or abdominal bleeding, or injuries to the extremeties may be also associated with the head injuries.

I realize that Dr. Coblentz' discussion did not bear upon the sequelae of head injuries, but I would like to ask him to say something about one of these troublesome sequelae—the so-called post-concussion syndrome.

PRESIDENT WAGNER: A great many of us are looking forward to hearing Dr. John W. Cline this evening in a prepared address. But since he is with us at this session, I would like to call on him now to make a few remarks pertinent to this subject.

DR. JOHN W. CLINE (President, A.M.A.): Thank you, Dr. Wagner. I have enjoyed this very comprehensive paper and the pertinent remarks of the discussants. I don't believe there is anything that I can add to the discussion.

When I was resident surgeon at the Bellevue—a good many years ago, we confronted often the difficulty, as one does in public hospitals of a patient who had been drinking and had a head injury of some degree. It was necessary to differentiate as to whether the principal thing was the alcohol or the principal thing was the injury, and I think in many instances that still holds true. I would be very interested to have Dr. Coblentz enlarge upon that, if he has time.

The other thing that we found out at that time at Bellevue, and the thing we have used in San Francisco County Hospital is to insist that the hospital doctor record periodically a fairly complete neurological examination, because of the changes taking place are extremely important and the memory of man is extremely fallible. If an actual record has been made periodically, then the changes can be demonstrated. And looking back over the past 25 years it seems to me the principal advance made in the treatment of head injury has resulted from a great understanding of the actual pathological and physiological changes they produce and the restriction of radical procedures to the elimination of those particular conditions, rather than in the old days, when the subtemporal decompressions were done for increased intracranial pressures of one type or another where there was no specific pathology which could be dealt with.

PRESIDENT WAGNER: Thank you, very much, Dr. Cline.

Is there any other discussion on this very interesting paper? If not, I shall call on Dr. Coblentz to close the discussion.

DR. COBLENTZ: I was very glad to have Dr. Gordy and Dr. Preston emphasize the importance of proper treatment of scalp wounds

and the prevention of infection, and also to emphasize the fact that all foreign bodies don't have to be removed, especially if it entails damage to brain tissue that is not adjacent to the actual damaged brain. Often a gunshot wound or fragment may be far displaced from the portal of entry. I have seen a lot of damage done in probing around even under x-ray, trying to take out a small foreign body at another point. I think judgment has to be used along those lines.

After the First World War there were plenty of foreign bodies left in, and the abscesses formed were all sterile, practically. Some had to be operated on.

The question of subdural hematoma in children I did not go into, because I think the treatment of cerebral trauma in children differs quite a bit from that in adults. They react differently. What might give an adult a headache, might give a child a convulsion. And with the proper control convulsive seizures I have seen the patient recover without any residual. That is not often seen in grown people.

The subdurals in children are very elusive. I remember a case of a child, a new-born, which had trauma at birth. The head started to enlarge and the child was referred to our clinic. We did subdural taps, the subdural fluid was slightly xanthochromic. And we felt the child had subarachnoid hemorrhage. We did tapping until the blood had almost cleared. In the meantime the baby's head kept enlarging. We couldn't act, for the reason there was nothing in the subdural space over the hemisphere and that the spinal fluid had practically cleared of blood. Through a very small incision in the scalp and a small opening in the bone we came down on the chronic subdural hematoma. The child was two weeks old by the time we got to it. It was a rare case and not often found in life but is found at autopsy. In fact, that was the only case I could find that had been found before death. There had been subdurals around the chiasm and around the base, but I think this was the only one in the posterior fossa. So it can happen there, but it is not frequent.

Dr. Cline brought up the question where you don't know whether the patient is drunk —you smell alcohol on his breath and evidently he has had a blow on the head. We treat the patients just as if they had never had a drink of liquor.

And probably a little more obvious and one of the things I didn't emphasize was a record on the chart of the progress of that patient, state of consciousness, paralysis, or any change in pulse or temperature, and that should be recorded, because in case of internes or nurses changing you just don't know, otherwise. Not only are you confused with those that come in with alcohol on their breath, but the patient may fall suddenly and strike his head and he comes in with a hypertension, at least you feel that he has such. You may not see him for two or three hours after the fall, and the increase in blood pressure may be due to the increase in intracranial pressure. That can be confusing at times. You can't tell whether there is a spontaneous hemorrhage or he had a stumble, or that the condition was due to cerebral trauma.

Way back some time ago we used to do spinal punctures of every case where we smelt alcohol on the breath, hoping that would bring him out of the alcoholic state, but you get into trouble doing spinal punctures on cases where that shouldn't be done. So our method is more observation on the fellow you feel is drunk and has had too much alcohol.

THE MEDICAL MANAGEMENT OF EPILEPSY*

DOUGLAS T. DAVIDSON, JR., M. D.,**
Boston, Mass.

Epilepsy, that condition characterized by periodically recurrent fits, spells, convulsions or seizures, affects at least 800,000 persons in these United States. Epilepsy affects more individuals than cerebral palsy, infantile paralysis or active tuberculosis. Like infantile paralysis, epilepsy strikes down its victims in all classes of society and in all age groups, though three-quarters are first affected as children. Like infantile paralysis, it strikes strong as well as weak. Every business, trade, art or profession contributes its quota of sufferers from epilepsy so that the handicap, obviously, does not preclude superior intellectual or artistic endowment or achievement. And yet, what is too often the

* Read before the Medical Society of Delaware, Wilmington, October 9, 1951.
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epileptic's place today? Stigmatized, isolated from normal social contacts, denied educational opportunities and all too frequently refused a chance to earn a living, he is driven to unwilling dependence on impersonal charity and is supported in idleness at the cost of millions of dollars yearly. At a very reasonable financial outlay to provide modern treatment and proper job training and placement, the majority of employable epileptics —two-thirds of all the adults—can be made productive members of society at a tremendous saving in dollars and with inestimable benefit to our society.

When the facts about epilepsy are known to a reasonable number of our citizens, new and more rational attitudes on the part of the public will prevail and the toughest part of the problem will have been solved. At this point I would like to advocate support for the National Epilepsy League, a lay organization which is pioneering on the fight to stamp out superstitions and misconceptions about epilepsy by making the facts available to all. I fully acknowledge our indebtedness to Jackson, Gowers, Berger, Lennoe, Penfield, the doctors who, through their ingenuity, industry and devotion have contributed so brilliantly to our understanding of this age-old scourge and have made possible the magnificent advances in treatment. Finally, I would like to pay tribute to those parents of epileptic children and to those individuals who suffer from the disease who are courageously and clearly demonstrating to the world that epilepsy is not a mysterious, loathesome and almost supernatural condition, but only another one of life's many handicaps to be faced squarely and carried gallantly—not a catastrophe, certainly not the end of all hope for a full life.

Today, we physicians can determine the cause or causes of a patient's seizures and prescribe treatment that will materially decrease or eliminate seizures in eight of ten epilepties. However, the most satisfactory results are not always achieved at once and the adjustment of medication may require several months or, in some cases, several years. Even in those patients whose seizures are not controlled after prolonged treatment, medical progress and particularly in the case of children, time works for their advantage. In view of this favorable outlook for the overwhelming majority of epilepties and yet with the realization that the problem of living with occasional seizures will have to be faced by those whose seizures cannot be immediately controlled, we physicians have the additional responsibility of teaching patients how to live with their seizures while carrying on as full and productive a life as their natural endowments permit. Stopping a person's seizures is of little use if he has missed the chance for education for learning how to get along with people, if he has been forced into considering himself an invalid—a rejected and worthless member of society. We must spare no effort to support the patient's morale—to keep him swimming in the normal current of life.

What are the strictly medical services which his physician can offer the seizured patient? Obviously, the first step is accurate diagnosis of the cause or causes of the attacks. I shall not discuss in detail the careful investigation of the nervous system for evidences of damage, nor the inquiry into the family history for evidences of a predisposition, nor yet the estimation of the role of emotional factors in precipitating the seizures. I would like to point out, however, that only one laboratory examination—the electroencephalograph, an electronic amplifyer which makes a record of the brain's electrical currents—is capable of suggesting the diagnosis of epilepsy. X-rays, chemical determinations of the blood and even surgical exploration of the brain may disclose no abnormality and yet the electroencephalogram, abbreviated EEG, may demonstrate the characteristic abnormality with startling clearness. Only in the past few years, since 1945, has the electroencephalograph become widely available as a practical clinical test for the assistance of the average doctor. The electroencephalogram is useful in differentiating epilepsy from fainting or hysteria, in locating brain injury or tumor, in indicating the seriousness of the case, in suggesting the best drug to be used and in following the success of treatment. The electroencephalograph is to the treatment of epilepsy what the electrocardiograph is to treatment of heart disease.

Having made a diagnosis of epilepsy and evaluated the etiological factors, the physician

then prescribes one of the six to eight medicines currently available for treatment of epilepsy. In general, the drugs can be divided into two groups—the first group composed of tridione and paradione, is effective against the petit mal type of seizure. Petit mal seizures, you will recall, are of three types—the absence or simple loss of consciousness lasting from five to 15 seconds with eyes open and staring or blinking, recurring many times during the day and usually affecting children. The second type in the petit mal group is the akinetic or drop seizure in which the patient instantaneously falls limply forward if sitting or to the floor if standing and recovers immediately. The third pattern of the petit mal group is the myoclonic jerk seizure which, in adults, usually involves one or a pair of extremities and, in infants, involves the whole body in a momentary shock-like contraction of the extensors or the flexors, producing a jacknifing or hyperextention of the body. In the younger age groups, these shock-like contractions may recur at 15 second intervals for a series of five to a dozen or more jerks.

The second group of drugs is useful in treating both the convulsive and the epileptic equivalent types of seizures. In this group are phenobarbital, mebaral, dilantin and mesantoin. The long established bromide is useful occasionally when other drugs fail but is generally too sedative in comparison with its anticonvulsant action. A new hydantoin, thiantoin, produced by Lilly, is said to be effective in this group of seizures, but recent experience has shown it to be toxic for the liver. A significant advantage over dilantin to warrant the risk. In the convulsive group of seizures is the generalized convulsion or grand mal and the focal convulsion involving only one extremity or one side of the body and that particular type of focal convulsion in which there is a gradual spread or march of symptoms—the jacksonian seizure. Epileptic equivalents or psychomotor seizures are characterized clinically by a trance-like state in which the patient often makes chewing or swallowing motions, drools and performs automatic movements if conscious though he has amnesia for the entire affair and often experiences headache or drowsiness following the attack.

The electroencephalogram may be extremely helpful in distinguishing between the petit mal type of seizure and the epileptic equivalent, or psychomotor type, for in the petit mal group the spike-wave complex spells the need for tridione or paradione and in the psychomotor group the spike discharge in the temporal region calls for the use of one of the hydantoins—dilantin or mesantoin as a rule. The electroencephalographic demonstration of a spike focus any place on the scalp may indicate the guilty area which is producing the sparks which periodically set off the generalized conflagration or convulsion.

In the following table, I have listed the most commonly used drugs and their top normal dosages.

Seizure Pattern		Age 1 yr.	Age 6 yrs.	Age 12 yrs.
Convulsive	Phenobarbital	2gr.	3gr.	3gr.
	Mebaral	3gr.	4gr.	6gr.
Psychomotor	Dilantin	1½gr.	4½gr.	6gr.
	Mesantoin	1½gr.	4½gr.	7½gr.
Petit Mal	Tridione	10gr.	20gr.	50gr.
	Paradione	10gr.	20gr.	50gr.

We usually start with about one-third to one-half the maximum dose and increase it, if necessary, and if tolerated by the patient. The total daily dose listed in this chart is given in several increments, usually after meals and at bedtime.

Tridione, paradione and mesantoin—three of the most valuable drugs for the treatment of epilepsy—are each of them capable of depressing the bone marrow, the most vulnerable elements of which are the neutrophils or polys and the platelets. It is, therefore, mandatory that blood counts be done at regular intervals on all patients receiving the newer drugs. We recommend a monthly white blood count and differential smear from which examinations the absolute neutrophil count can be calculated and the presence or absence of a normal number of platelets on smear noted. If the absolute number of polys is depressed to 2500 per cubic millimeter, the blood count is done at fortnightly intervals; and if the depression of the neutrophils reaches 1600 per cubic millimeter, the drug is stopped. We expect a return to normal in three or four days. If the neutrophil count falls below 1000 inadvertently, then we rec-

ommend the use of vigorous antibiotics therapy in order to prevent bacterial invasion of the system during the period of lowered body defenses.

All the anti-epileptic drugs are capable of producing a rash in certain individuals and mesantoin seems to be the worst offender in this connection. Fever and lymphadenopathy often accompany the measles-like rash of mesantoin. Failure to stop medication promptly in the presence of a rash from any of these drugs may result in exfoliative dermatitis and possibly in aplastic anemia or massive liver necrosis. The anti-histaminics will stop the itching which often accompanies the rash, but usually does not hasten the disappearance of the eruption which spontaneously fades in from 24 to 96 hours.

Painless swelling of the gums is produced in possibly half the patients who take dilantin, but rarely necessitates discontinuance of the drug. The only treatment which seems to help is faithful and skillful massage of the gums by the use of a toothbrush. In severe hyperplasia surgical excision of the excess gum tissue may be required, but this promptly recurs unless massage is carried out as soon as the gums heal.

Some degree of sedation is produced by massive doses of nearly all the anti-epileptic drugs, but especially by phenobarbital. Dilantin has the least sedative effect of all, but in young children produces vomiting in excessive doses and in adults ataxia, nystagmus or ambliopia.

We feel that medication should be prescribed continuously with a constant daily dose once the effective dosage is discovered and it should be maintained until at least six months after the last seizure and at that time, if the electroencephalogram has cleared up, the dosage may be cut in half gradually. Six months later, if seizures have not recurred and if the electroencephalogram is still normal, the other half of the medication may be gradually withdrawn.

It is important to explain to the patient at the beginning of treatment that the initial prescription is the one which is most likely to succeed in his type of seizure but that because his individual reaction cannot be predicted, other medicines may have to be tried. Rapport is usually improved if the patient is made a partner in the trial of medication, the doctor suggesting the drug and the dosage and the patient evaluating his reaction to the drug and the effect upon his seizures. Only one drug should be changed at a time and the change should be gradual enough and continued long enough for accurate observation of effects.

In summary then, we have discussed the magnitude of the problem of epilepsy and its unique position among handicaps posed by the stigmatization associated with it. We have outlined recent advances in diagnostic and treatment methods which if employed on on a wide scale could revolutionize the outlook for the overwhelming majority of our three-quarters of a million epileptics.

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DISCUSSION

DR. A. L. INGRAM, JR. (Wilmington): I would like to congratulate Dr. Davidson on a very stimulating and succinct presentation of an admittedly difficult subject. With the increasing strides and advances made it becomes increasingly more difficult to present the essential facts in an understandable form, and I think this has been accomplished most admirably this morning.

I was happy to note that Dr. Davidson made a statement concerning the teaching of patients to live with their seizure. As he noted, it often takes many, many months, even years, to be able to find the optimum dosage of the correct drug to control the seizures of the particular patient. It is during this time that psychotherapy can be most useful. However, all too often we neglect the psychotherapeutic aspects in the treatment of the seizure patient, which often gives rise to preventible psychiatric problems. Too often we are prone to prescribe a drug, tell the patient how to take it, and sit back and renew prescriptions. It would take but little more time to reassure the patient, to give him some understanding into the disease process, and some insight, and to have him thus reassured of being, as suggested, a "partner" in the effort to control his disease. This is probably no more true anywhere than in the problem of epilepsy for it is there that we are treating an individual as well as a disease.

I have one question I would like to ask Dr. Davidson, that is, in regard to the use

of tridione and paradione in the treatment of the petit mal epilepsy—whether or not the one has been considerably more effective in controlling this type of epilepsy than the other in his experience. We have found relatively little success in the use of tridione and have had a very limited experience in the use of paradione.

DR. M. A. TARUMIANZ (Farnhurst): I wish that the term "epilepsy" would be discontinued from our medical vocabulary. It is a misnomer. It doesn't state the fact that epilepsy, per se, is not a one-entity, or a one-type of disease. Therefore, Dr. Davidson in his presentation placed emphasis on the fact that the establishment of the diagnosis of convulsive seizures is a most important factor in the establishment of the treatment of such cases.

I believe if we consider very seriously that many children as well as adults have convulsive seizures and do not have epilepsy, per se, and that convulsive seizures are due to some other factors, diseases of the brain, the central nervous system, which can be diagnosed adequately with electroencephalogram studies, which has been a new tool in the hands of doctors, we must realize that we are very fortunate in this stage. For the past few years we have established a department for convulsive diseases at the Governor Bacon Health Center. It is your center in this state. Every physician has a right to refer a case for such study and a report will be immediately sent to the referring physician. In that way you can obtain a knowledge as to the cause or causes of the convulsive seizures—whether the individual has severe damage to the brain or the convulsive seizures are due to other factors which one did not think about at the time of considering the therapy.

I wish to again emphasize the fact that there is no reason for anyone to send a patient to any place outside of this state. We have a laboratory which is the State Laboratory. There we have the best facilities and well-equipped technicians under the guidance of a Philadelphia expert who assumes the responsibility of interpreting those tracings.

Again I wish to congratulate Dr. Davidson for his paper, as well as to invite him to stop at the Gov. Bacon Health Center and see the Department for Convulsive Seizures. And I should like to urge, while the President of the A. M. A. is here that we do something about changing our approach to convulsive seizures and try to eliminate the term "epilepsy", which has certainly done more damage to the emotions of our people than anything I know of. It is a sad story to anyone who has practiced medicine as long as we have—some 47 years—to realize what it means to the family, the children or adults who are handicapped with these "curses" as accepted by our nation, and, as a matter of fact, by humanity.

DR. R. E. REED (Delaware City): I have been on the staff of the Gov. Bacon Health Center for about two and a half years. I would say that the greatest advance that the state of Delaware has added to the science of epileptology is the concept, which as far as I know originated with Dr. Tarumianz, that a child with epileptic seizures is primarily a behavior-disturbed child, a maladjusted child, and that he belongs first probably in the province of the psychiatrist.

Of 175 mal-adjusted children we have there are about 75 in the epileptic children's service. They go on walks and play baseball, they go to the show, they go to the drama; when the children go off grounds they go to the horseshow and air show, they are brought to theatres and they go in our swimming pool. They do, in fact, everything all the other children do, and to look at them, and as far as they know, they are not any different from the other children there. And I think that is in most cases the first time in their lives they have been treated in such a way, and I suspect somehow such treatment is related to the fact that they don't seem to have any more convulsions.

Another thing we learned in this connection is that many of them are slightly undertreated by their doctors. The profession is becoming farther up-to-date in the drug treatment of epilepsy than previously. I know it all too well, because I look back with disgust on my early experiences and of course our training was always to use as little medicine as we could get away with. We have learned to use greater doses and then grudgingly and slowly reducing it down to the minimum, rather than to allow the child to

go on with just too little medication and keep on having spells.

DR. DAVIDSON: The question about triodione and paradione. As to that I might say we couldn't run an epileptic clinic without these two drugs. We might be able to run an adult clinic where we would see very little petit mal, but in children the occurrence of petit mal runs between one-quarter and one-third of all our patients in one form or another, and without those two drugs we couldn't do a proper job.

Dr. Tarumianz has brought up a very real problem, the "sense of guilt" for the child's seizures, which completely warps the parent-child relationship. This sense of guilt is a very real one—it is not to be side-stepped by changing the name of the disease or by pretending that there is a more euphemistic name for it. But the question of guilt must be faced according to the reason for the parents' need and by educating a parent according to the modern concepts of a symptom complex.

We do not say that epilepsy is a disease: far from it. It is a symptom like headache and, like headache, has an infinite number of causes. The seriousness of the headache is no guarantee as to the seriousness of the cause. There is no correlation as to the seriousness of the cause, of course.

About Dr. Reed's comments. I am very happy to have him point out the importance of the emotional factors in precipitating convulsions in other types of seizures. The emotional factors may be all-important in many patients. They may be practically non-existent in others—but I think in every patient that we are evaluating we must take recognition of the fact that their seizures may not be only tremendously aggravated, but even impossible to control, unless we make some provisions to take care of the emotional disturbances which are present in that youngster. We are indeed fortunate, here in Delaware, in having a Center like the Gov. Bacon Health Center, where patients can be referred to electroencephalographic diagnosis. Such a diagnosis, in my opinion, for any patient that doesn't respond immediately to medication is imperative—or such an examination, is imperative. We can't treat patients intelligently without it, and we are very fortunate in Delaware to have these facilities available.

I think we are also lucky here in having a place like the Health Center, where children who are too ill to be handled at home can be referred and taken care of adequately. In some states there is no place to send such patients where there is too much strife or emotional upheaval in his home. Delaware is lucky to have such an institution.

CERTAIN ASPECTS OF SURGERY OF THE NEWBORN*

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To those who interest themselves primarily in the surgical problems of infancy and childhood, there seems to be no end to new ideas which can be developed in the operative and post-operative care of children, which lead to smoother convalescence, decreased morbidity, and lower mortality rates. In the field of pediatric surgery, however, the challenge still lies in the surgical management of those congenital anomalies which are incompatible with life, but which are amenable to surgical correction.

There are four such problems which might be included in this group without question. They are omphalocele, atresia of the esophagus with or without tracheoesophageal fistula, atresia of the bowel and imperforate anus. To this group I would also add meconium ileus and diaphragmatic hernia. Meconium ileus should be added because of the distressing mortality this problem carries with it about the country, and diaphragmatic hernia because its mortality, although not one hundred per cent, is so close to it as to be included in this series.

The management of omphalocele was moderately efficient as outlined by Ladd and Gross[1] some years ago, but an even superior method is that described more recently by Gross[2], wherein the membranes of the omphalocele are not dissected free, but rather are covered by skin which can be loosened from surrounding structures and stretched over the surface of the membraneous covering of the abdominal viscera. We have made but one slight improvement on this technique. In

* Read before the Medical Society of Delaware, Wilmington, October 9, 1951.
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large omphaloceles where disproportion between the protruding viscera and available skin is great, it is possible to compress the abdominal viscera by rolling the membraneous covering in much the same way that one would roll down the top of a paper bag after bringing the two sides together, and then to approximate the skin edges while holding the viscera, thus tightly compressed. Omphaloceles extending from the xiphoid to the pubis, which otherwise might not be correctible by surgical means, can be closed in this fashion.

In reference to problems of atresia of the bowel, recovery still seems to be proportional to the age of the patient, the surgical care exercised at the time of operation, and the excellency of post-operative care.

Meconium ileus, diagnosed early by the signs of vomiting, distention, viscid secretion, and shadows resembling feces on x-ray examination of the abdomen is being successfully treated insofar as the obstructive problem is concerned by a variety of surgical approaches with a decreasing mortality. Whether one performs a temporary ileostomy or resects the affected portion of the bowel, the use of pancreatic enzymes and preserved duodenal juice are of great help in establishing the passage of stools.

In the treatment of diaphragmatic hernias, we have abandoned the abdominal approach and routinely operate transthoracically.[3] In the thoracic approach it is possible to open the chest of a distressed infant in a matter of a minute or two, and to eviscerate the abdominal organs from the thoracic cavity, at which moment the baby becomes a satisfactory patient from the point of view of cardiac and respiratory reserve. The collapsed lung can expand, the heart is no longer compressed, and the mediastinum may shift back to its normal position. The surgeon need then be in no hurry, but can at his leisure replace the abdominal viscera into the peritoneal cavity through the diaphragmatic defect; the chest is then closed in the usual fashion after suturing the diaphragm, and the infant returned to his crib with a chest wound which he seems much better able to handle than a large abdominal wound behind which are more abdominal contents than the peritoneal cavity can conveniently accommodate. We have done fourteen diaphragmatic repairs by this technique with one death, and that in a child with premature ossification of the skull and subdural and epidural hematomas.

Having thus disposed of four problems with little more than a word, I would like to turn our attention to the two remaining problems: atresia of the esophagus, and imperforate anus, and outline a method of management for each which we have found satisfactory and which in each instance seems to be somewhat at variance with the usual techniques employed.

In the management of atresia of the esophagus our earlier method of management was that used by most, a retro-pleural approach through the posterior mediastinum, at which time the tracheoesophageal fistula was ligated and an end-to-end anastomosis carried out between the blind pouch above and the open esophagus below. On the day after this procedure a gastrostomy was done, feedings were delayed for a period of five to ten days, and frequently post-operative dilatations of the esophageal anastomosis were necessary in order to permit the passage of food through the sutured esophagus. This method of management had many faults. The operative procedure was long, it was difficult to stay out of the pleural cavity, the hospitalization was prolonged, and economic loss to the family was great. There was always the risk of regurgitated gastrostomy feedings, the need of two operations, and the delayed nutrition of the patient. Finally, the problem of esophageal dilatations and the occasional necessity for surgical closure of the gastrostomy was time-consuming. In addition, the patients frequently developed edema post-operatively, which in those children who died, at times approached a generalized anasarca. Under this technique we felt that we had done all that we could to improve our management, and the mortality was over fifty per cent.

At the present time, we have altered our method of caring for these children in this manner: instead of using a retro-pleural approach, we operate transpleurally and reduce the operative time from a period of two to five hours, to an average operating time of an hour and fifteen minutes. No gastrostomy is carried out following operation if we have reasonable assurance that the suture line is tight. On the morning of the second post-

operative day the patient is fed glucose water and his formula is increased rapidly to something similar to that given to most newborn infants. By this method, not only is anesthesia and operating time decreased, but only one operation is necessary, hospitalization is shorter, the economic loss to the family is less, nutrition is maintained, and surprisingly enough, esophageal dilatations do not appear to be necessary as frequently as heretofore. Edema is now seldom seen because we maintain these children on no salt (except that contained in blood transfusions) for the first three post-operative days, and then limit fluids to approximately 60 cc./lb/day.

If one excludes patients with a second anomaly incompatible with life and patients with multiple congenital defects, which in themselves carry a high mortality, the mortality is surprisingly low. Out of 20 such patients we have had 15 survivals. Four gastrostomies were done in this group and two patients needed dilatations of the esophagus. This method seems to us to be a rational approach to the problem of atresia of the esophagus, not only in the hands of the pediatric surgeon, but also in the hands of the thoracic surgeon who only occasionally deals with such problems. The risks are less, the procedure easier, and the results better.

The problems resulting from the improper management of imperforate anus have long been a burden to us. Because this is the most common of the congenital anomalies incompatible with life, a large number of surgeons encounter the lesion and operation is carried out by many unfamiliar with the end results of this variety of surgery. Most patients with imperforate anus are divided into two types: Those in whom the blind rectal pouch can be reached from below, and those in whom the distance between the anus and the colon is too great to be bridged by a perineal operation, in whom a colostomy seems the procedure of choice. This is a satisfactory classification of patients, but the selection of operative procedures for each is frequently unwise.

First, and most dangerous, is the common attempt to bridge too wide a gap between anus and colon by the perineal approach. When such is done it is possible to bring colon mucosa to perineal skin for a suture line, but this is done at the expense of nerve and blood supply to adjacent areas. Such suture lines, under great tension, do not hold, and there develops in a matter of days after operation a rectal canal surrounded by fat and muscle and lined with granulation tissue. This in turn becomes a tight, relatively long stricture which can make the patient a rectal cripple for the rest of his days. Not only are these strictures very difficult to correct but frequently their correction leads to incontinence. If they are not corrected, obstructive megacolon may ensue.

The second variety of mistake is made in those patients in whom a colostomy seems necessary. The colostomy is carried out in the sigmoid colon, thereby utilizing the redundant loop of colon which should be left free to provide bowel to construct pelvic colon, rectum, and anal canal at the time of definitive operation. These two errors can be eliminated by never attempting to bridge a gap between colon and anus which is greater than 1.5 cm., and when a colostomy seems indicated, by placing it in the right side of the transverse colon in order to leave as much distal colon as possible for use at the time of definitive operation.

We attempt to divide all of our imperforate anus cases into three groups as to operative procedure. First, patients with a blind gap of 1.5 cm. or less are approached through the perineum, and an anus constructed which usually has some sphincter. Secondly, patients who weigh less than four and a half pounds, or who present some other medical problem which contraindicates major surgery, have a colostomy performed in the right transverse colon. These two methods could be carried out satisfactorily in almost any community where surgery is practiced.

At the Children's Hospital of Philadelphia, we have added a third category of operation,[4] believing that we have sufficiently good anesthesia, nursing, and house officer care to warrant such type of work. Those with a gap between anus and colon of more than 1.5 cm. have a definitive operation carried out at the time of admission to the hospital. A combined abdomino-perineal type of approach is carried out with the abdomen being opened first and the blind end of colon secured and exteriorized. The patient is

draped in such a way that one leg is outside the drapes and in the operative field. A small section of skin and skin only is excised from the region of the anus, the external sphincter cleanly divided, and a small hemostat is inserted into the perineal body. This aperture is gradually enlarged until it is possible to reach up into the true pelvis, and bring down through the perineal canal thus made the blind loop of colon and exteriorize it through the newly formed anus. These children are taken care of once and for all, and the only thing that remains to be done to them is to have the excess colon trimmed off by a very minor procedure just before discharge from the hospital.[5]

Patients of three days of age or less tolerate this extensive surgery without difficulty. It is of interest to note, however, that if such an operation is not carried out in this very early neonatal period, it becomes one of the most shocking procedures one can undertake in pediatric surgery, and therefore, by experience we have learned that if this cannot be accomplished early, one should do a colostomy and then at a later date, after the age of three or four months, perform the definitive operation by the combined abdomino-perineal approach just described. When patients are cared for by the three techniques outlined there should be no rectal strictures. Tight anuses can be dilated in infancy, resulting in a cosmetically acceptable anus, and a well-functioning lower bowel. Such patients take longer to train for stool habit than normal babies, but they usually can be made into satisfactory citizens from the point of view of bowel function.

I will not go into the many problems of fistulae between the colon and the urinary tract, which so frequently complicate this congenital defect. They can be managed satisfactorily during the procedures outlined. We have operated on almost forty of these youngsters in the last four years with one death and one failure. Several premature babies have died from other congenital anomalies before surgery was undertaken. The smallest baby we successfuly treated weighed two pounds, twelve ounces. None are now rectal cripples nor need look forward to that existence in the future. In the same period of time we have treated a large number of patients secondarily who had previous surgery at the time of birth, surgery which was not carried out according to the principles here stated. We think that the proper management of such patients could eliminate the very serious problems we see of fecal incontinence, megacolon and rectal stricture following surgery of imperforate anus.

We believe that much can yet be done to improve the technique of operative procedure and post-operative management of patients with atresia of the esophagus and imperforate anus. We realize that a low mortality rate in a small series of patients may be misleading and that we have had a particularly good succession of patients in both categories. However, we do believe also that some of the improved mortality is due to careful attention to detail based upon correctible errors of the past, and suggest that the method of management outlined for each of these conditions warrants trial in other pediatric installations.

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DISCUSSION

DR. R. O. Y. WARREN (Wilmington): I am somewhat amazed to be asked to discuss a surgical paper. Dr. Koop is always a welcome guest to Wilmington. I, personally, would like to thank him for the courage he has displayed in these problems. As a mere pediatrician I cannot cope with the technical detail of Dr. Koop's paper, but I would like to point out one or two things that are somewhat alike or of a kindred nature, and that I think are equally important.

One is that anybody in this room or anyone in the state of Delaware, in fact, that has anything at all to do with young or newborn babies may see some similar condition in an hour, a night, next week, or maybe not for the next twenty-five years.

I see one confrere of ours sitting in the room that went an extraordinary length of time before he saw a condition akin to that

of which the doctor has spoken, so the point I would make is that those general practitioners, pediatricians, obstetricians—anyone having to do with newborn babies—have to be on their guard and "on the ball". And from there on it is, in our community, at least, a team effort.

Dr. Koop makes it seem very simple—the surgeon just gets the baby to the operating room and he promptly and very expertly takes care of him! That doesn't say, of course, that there hasn't been a lot of preliminary study in some of these babies, and it doesn't say that the baby hasn't been prepared for operation, and isn't cared for afterwards.

So the other point that I would make is that in an average community such as ours the management of a case such as this, beside the expert skill supplied by the surgeon, is a team-effort in which a great many other people are involved. Strangely enough, I don't think we were all blind or completely dumb, consequently this did not seem to occur so frequently before the war as since. And we feel greatly interested in this, as pediatricians, and some of our surgeons here in town have become interested in this condition, too, and everything that Dr. Koop has said on a previous visit and today I feel is most worthwhile and extremely valuable.

DR. H. H. STROUD (Wilmington): From the viewpoint of the practicing physician, earlier diagnosis of, and earlier treatment of the conditions mentioned by Dr. Koop will further decrease the too high mortality.

The omphalocele is obvious by inspection at birth. Too frequently operation is delayed until the integrity of the covering membrane is lost; then infection and peritonitis increase morbidity and mortality.

Esophageal atresia can be diagnosed when water is offered as the first feeding 8 to 12 hours after birth. After a few swallows the infant with atresia will cough and choke. The diagnosis is established by simply introducing a soft rubber catheter which will meet obstructions after being passed only a few centimeters. X-rays are unnecessary in most instances before referral to the surgeon. Too frequently such is offered the baby with atresia over a period of days before the diagnosis is made. Then the surgical problem is complicated by a lipoid pneumonia.

Meconium ileus and atresia of the intestine present similar symptoms. Vomiting occurs within a few hours to a day after birth, associated with abdominal distension and frequently peristaltic patterns can be seen on the abdominal walls. The diagnosis can be confirmed by instilling a few cc. of iodized oil in the stomach to observe its passage to the point of obstruction under the fluoroscope.

With diaphragmatic hernia respiratory distress is an almost constant finding. An early chest x-ray will reveal the underlying difficulty where the physical findings are in doubt or obscure.

Hence, the procedures required for the diagnosis of these congenital anomalies are simple and usually available to all. If the conditions are thought of, diagnosis is easy within a day or two of birth. The surgeon can operate when conditions are best and morbidity and mortality will be reduced.

DR. KOOP: In trying to keep within the time limit and cover the several points that I was particularly anxious to present in my paper, I want to make it clear that I didn't attempt to stress the diagnosis of these problems, and I did not mention the cooperation necessary for the care of these patients both before and afterwards with our pediatric confreres. I want to thank the doctors for completing the general problem that I tried to present to you.

I would like to stress several things which can not be emphasized too often. One is that the newborn infant has its mother's physiology for about three days, and during that time he is a better operative risk than he will be at any time for at least two weeks, and if one plots mortality against age, the mortality is about steady in the first 70 hours of life and then by the eighth day of life the mortality is so close to 100% as to be equal to it.

I would like to caution those having to do with the preoperative problems not to use barium. One thing that makes for difficulty is having barium in the tracheo-bronchial tree. Children having the problems mentioned here tend to vomit those things given by mouth.

Finally, although we have a pediatric-

surgical service of surgeons who are trained somewhat pediatrically and manage their own problems to a great extent, in many other hospitals throughout the country surgeons have to do the technical work and pediatricians have to take care of the child's intake. We have found generally that almost all the patients need more fluid post-operatively than does the ordinary child, but can get by with less salt.

Finally, much of the success of the pediatric endeavor is due to the anesthesia available. The mortality, as low as it is in this surgery is due in great part to Dr. Margery Deming and her staff at Children's Hospital.

FICTION AND FACTS ABOUT LIPOTROPIC MEDICATION IN ATHEROSCLEROSIS

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In want of a proven weapon against atherosclerosis, patient, physician and pharmacist reach out for any therapeutic measure described or suggested. They comb the literature for any hint of prevention, arrest, or successful reversal of vascular disease in man or in a laboratory animal.

It is a fact that rabbits and other rodents with induced prolonged or severe hypercholesterolemia commonly develop vascular alterations resembling human atherosclerosis. It also is a fact that prevention of hypercholesterolemia in a cholesterol fed rabbit will result in the prevention of cholesterol-atherosclerosis. It further is a fact that with spontaneous or artificial reduction of the rabbit's hypercholesterolemia to a normo-cholesterolemic state initial vascular lesions will undergo reduction in number and in degree.

It has been stated that rabbits with induced hypercholesterolemia can be protected from atherosclerosis by increasing plasma phospholipids. This is not a fact. The assumed preventive action of phospholipids has been ascribed to its ability to stabilize colloidal cholesterol. This is fiction. Phospholipids, namely lecithin, have a modest stabilizing effect on cholesterol sols *in vitro,* but no effect *in vivo.*

There is a mathematical relation between blood cholesterol and phospholipids in animals as in man. This ratio, whether expressed in milligrams or in milliequivalents per blood volume is altered in experimental animals with induced hypercholesterolemia, namely in chickens, along definite patterns. In patients with clinical manifestations of atherosclerosis this ratio is as often normal as it is altered. For that matter, hypercholesterolemia per se is as often absent as it is present in these patients, and where it exists it rarely approaches the degree noted in animals on a cholesterol diet. Moreover, in man, the phospholipid level fluctuates, as a rule, with the blood cholesterol level. Thus, it seems ill advised to promote therapeutically an increase of blood phospholipids, in human beings.

Still, for some time, various compounds known as lipotropics have been given to animals and human beings. Such therapy was and is based on the fictitious reasoning that it will regularly reverse the proportion between blood cholesterol and phospholipids and that it subsequently will influence atherosclerosis.

It is true that many substances enhance the phosphorylation of lipids: monomethyl-ethanolamine, dimethyl-ethanolamine, and ethanolamine alone, all are more effective in this respect than choline. While choline increases choline-containing phospholipids only, ethanol increases all phospholipids in the liver. The relation between the rate of lipid phosphorylation in the liver and the amount of lipotropic factors in the diet is not a simple one. As a matter of fact, all diets contain sufficient quantities of methionine, choline and ethanolamine for phospholipid synthesis. The crux of the matter does not lie with phosphorylation as such but with transmethylation. The dietary supply of transferable methyl groups represents a limiting factor for the synthesis of phospholipids in the liver. The formation of phospholipids in the liver becomes deficient where there is an increase in dietary substances competing for the methyl group (for example, guanidoacetic acid) or acting as metabolic antagonists of the methyl reception in the synthesis of choline (for example, diethanolamine).

The various lipotropics do not act in like

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manner. Phosphorylation is enhanced if the medicated patient is on a low protein diet but inhibited by a high protein diet. Vitamin B 12 affects transmethylation more than other substances. The activity of inositol depends upon the absence of dietary fatty acids. Addition of choline to a high fat diet results in an increase of hepatic lecithin; choline added to a low fat diet has no such effect.

It is a fact that lipotropics affect the phospholipid content and the total fat content of the liver. Their administration results in an increased turnover of hepatic phospholipids, namely of lecithin. That they affect the blood lipogram is a fiction. To measure the efficacy of lipotropics by the cholesterol or the phospholipid blood level is impossible. To gear the medication to the ratio of the two blood lipid complexes is erroneous. In about one third of patients on lipotropic therapy the blood lipids remain stable, in roughly one third, the lipids decrease and in about another third there is an increase. Regardless of the dosage or of the length of treatment in man or animal, the cholesterol-phospholipid ratio is as often stable as it is altered in one direction or the other.

Reports that experimental atherosclerotic lesions induced by feeding a high cholesterol diet can be prevented or resorbed by choline or inositol belong in the realm of unadulterated fiction. The observation by one investigator that the mortality of patients treated over a three year period with choline after their first attack of coronary thrombosis and myocardial infarction was only forty per cent that of a control group not medicated with choline does not license another author to conclude that this mortality reduction was due to a reversal of atherosclerotic alterations resulting from the removal of vascular cholesterol by choline. Lipotropics do not remove cholesterol from the blood vessels. That's a fact. Neither do they remove cholesterol, free or esterified, from the liver of man or animals. That, too, is a fact. On the other hand, it is a chemically and histologically proved fact that lipotropics reduce the neutral fat content of the liver.

There is no doubt that many persons, especially those of advanced age, have functional and structural hepatic alterations. Many times, the hepatic deficiency is but slightly apparent or is non-apparent to the patient and the physician. Often, a battery of chemical tests including an electrophoretic blood protein assay are needed to detect the disorder. A liver biopsy might be required to diagnose organic diseases. Where the liver is damaged, administration of lipotropics is indicated. Such therapy furthers the feeling of well-being of patients, even so they are ignorant as to the nature of medication. The patient's subjective impression linked with improvement of the hepatic function objectively ascertained by sensitive laboratory studies are the criteria for evaluation of lipotropic therapy. The effect of lipotropics is transitory, only. The period of remission upon discontinuation of treatment varies from one person to another but will rarely exceed three weeks. Continuous treatment is warranted in these patients, or intermittent therapy with short intervals between medication, be this oral or parenteral.

The number of commercially available lipotropic products is large. Some products contain choline, others choline chloride, choline gluconate, choline dihydrogen citrate, or tricholine citrate. Some drugs contain choline combined with d-l-methionine and/or inositol. Still other products contain vitamin B^{12} as well. The concentration of the different ingredients varies from one product to another. The discrepancies in the recommended daily dose of these drugs are as wide as the variations in the proportion of the components. The proper combination and dosage could only be answered by systematic re-evaluation of all lipotropics. One would have to administer each substance separately in varying dose and try all possible combinations of the known parts of the lipotropic spectrum on large series of animals with hepatic steatosis of comparable degree. Clinical evaluation on human patients would have to follow.

Not infrequently we hear the question whether lipotropics are of true therapeutic value in patients with atherosclerosis. Since we lack knowledge of the true mechanism by which lipotropics act and since we are reluctant to repeat the various concepts just labelled as fictitious we are apt to avoid a direct answer to the question. One might reply that these drugs are harmless. It is true that no

side or ill effects have been reported from the use of lipotropics. That does not mean that no such effects were ever noted or that none will be observed in the future. The lipotropics are part of the vitamins. It took decades of widespread vitamin therapy to recognize the several hypervitaminoses.

Many manufacturers of lipotropic drugs add to choline, methionine, inositol and vitamin B^{12}, varying amounts of vitamin A, B^1, B^2, B^6, C, or E. Others also add rutin, calcium pentothenate, dextrose, thyroid substance, liver concentrate and desiccated liver. We are not certain that inositol by itself has lipotropic property, whether its activity is enhanced by choline, or whether inositol enhances the lipotropic action of choline. The addition of various vitamins and other substances further beclouds the picture and interferes with evaluation of lipotropics. This practice harbors some dangers because there are no valid criteria for dosage and discontinuation of therapy, especially as medication is left mostly to the patient himself. While the intake of thyroid substance—to select but one of the several ingredients encountered—is warranted in many aging patients, its administration certainly should be controlled by the physician.

The formulation of a scientifically supported concept of the lipotropic mechanism is most desirable. Such therapy is neither specific for atherosclerosis nor for hypercholesterolemia, or for hypophospholipidemia. However, it need not be supported by conjured reasoning. The reversal of hepatic steatosis and the improvement of the patients' well-being justify fully the usage of lipotropics, in spite of the lack of scientific criteria. Lipotropics should be prescribed for patients with atherosclerosis in whom there is evidence of liver impairment.

BLACK WIDOW SPIDER BITE

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Latrodectus mactans, the black widow spider, is found mostly in tropical and temperate zones of South and North America. In the United States it is found mainly in the southern states. The greatest number of patients bitten by the black widow spider, has been reported from California, but there are also reports of bites as far north as the New England states.

The female, which is most dangerous, is shiny black with spherical body, one to one and one-half centimeters in length, and has on the ventral surface an orange-red inverted hour glass marking. The male is much smaller, about one third the size of the female. In addition to the female markings, it has white stripes on the abdomen. Too, the male body is more elongated and has an orange dorsal stripe. When first hatched the female and male are identical in appearance, but later, during growth, they assume their respective characteristics. The male life span is shorter than the female. After fertilization of eggs, usually the female eats the male, especially when she is hungry.

The spider prefers dark, dry places, and is generally found close to human habitations. It also lives in woods, bushes, and fields. It likes protected places as under boards, under rocks, waste cans, shoes and clothes, and also across the seat openings of outdoor privies.

The web has no regular pattern, and also has a poorly developed funnel hiding area. During the daytime the spider usually remains out of sight in a protected corner. During the night or in darkness it is more active and is in open areas.

The black widow has a pair of hollow fangs connected to a pair of poison glands, which are located in the cephalothorax, just behind the eyes. The venom of the full grown female is more poisonous when she is distended with eggs. The poison sacs increase in size and the venom increases in strength as it grows to maturity. The entire body of the spider is reported to be inpregnated with the venom, even the eggs. The poison of this spider is more potent than that of a rattlesnake, taking equal parts.

The latrodectus mactans deposits in the bite a toxalbumin venom, a fraction of which produces paralysis of the ascending motor nerve endings. Gajardo states that latrodectus mactans affects mainly the central nuclei of the negative nervous system. He also thinks that the entire syndrome can be explained by the effect of the venom on the medulla bulb and the other parts of the brain. Several fatal cases are usually associated with

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pulmonary and cerebral edema, and persistant uremia with urinary retention.

The local reaction of the bite is slight. Usually there is a small slightly elongated pinkish spot with two insignificant openings. There is local or generalized edema. The syndrome of arachnidism, which follows the bite, has been observed in Milford Memorial Hospital during the past four years in five cases. One case was observed just ten minutes after the bite; other cases from two to nine hours after the bite.

The first case was a 21 year old white female waitress. When she put on a sweater one morning she felt an acute sticking pain in the left elbow region. At first she thought it was a needle, then she felt an insect. After removing her sweater she saw a spider. Ten minutes later she was in the hospital dispensary. Locally, on the left elbow region, there was a small urticaria-like skin elevation with two small bite dots. There was no pain at that time. About twenty minutes later the patient started complaining of cramp-like pains in upper arm and shoulder. Forty-five minutes after the bite the patient was complaining of pain in upper part of the back. About thirty minutes later she complained of severe cramp-like pains in her abdomen, especially in the epigastric region. At that time there was slight nausea.

The first examination was made upon arrival, but no significant findings, except the previously described local reaction. Second examination was about 20 minutes later, showing some tenderness in the upper arm. The muscles were more rigid on the left side than on the right. The neck and abdomen were entirely negative. The third examination was done about two hours after arrival. Temperature 98F, P 80, R 20, B.P. 110/70. Eyes: pupils equal; react to light and accommodation. ENT: negative. Neck: slight nuchal rigidity was present. Chest: There was muscular stiffness around axillary and pectoral region on the left side. In the back there was some soreness in scapular region and around the upper part of the thoracic vertebra. Heart and lungs were negative. Abdomen: not distended. There was some muscular rigidity, especially in the epigastric region, but very slight tenderness. Extremeties: the left arm was painful to move and muscles were slightly contracted. Neurologic: Reflexes were slightly exaggerated. She was complaining of slight headache. Laboratory tests three hours after bite: Urine: neg. Blood: RBC 4,900,000; WBC 7,050; HG 95%; color index .97; Schilling segs 61; lymphs 33; stabs 9: juveniles 1: baso 1.

The other four cases came to the hospital with fully developed symptoms. All complained of severe abdominal pain and of spreading pain from the bite side. A three year old boy was bitten on the penis in an outdoor privy. He was brought to the hospital nine hours later. He was very restless and the abdomen was board-like, resembling an acute surgical abdomen. A 66 year old white female had similar agonizing pain and abdominal rigidity. She was brought to the hospital two hours after being bitten on the left thigh, also in an outdoor privy.

The fourth of our cases was a 29 year old white man who was bitten on the neck six hours before admission while working in a garage. His chief complaint was severe pain in the neck and chest, and agonizing pain in the epigastric region.

The fifth case was known after the patient expired when additional history was given by the family. While not clear, it was suspicious, indicating that the patient had been bitten by a black insect in a privy about 48 hours before admission. This patient was 66 years old, white male, sick for several years with hypertension and anemia. He was admitted to the hospital in poor general condition. History was difficult to obtain, but his chief complaint was abdominal pain and cramps in lower extremities. Also, he had a headache and stiffness in the neck. On the death certificate the cause of death was given as uremia.

The study of the symptoms of black widow spider is very confusing. Many such cases are sent to the hospital as surgical emergencies, and the differential diagnosis must rule out the numerous causes of acute abdomen, but especially of ruptured peptic ulcer, renal colic, acute appendicitis, peritonitis, cholelithiasis, acute pancreatitis, food poisoning, and acute enteritis. The most confusing and the most important is differentiation from ruptured peptic ulcer. In ulcer cases usually there is no history of an insect bite, but there

is a typical ulcer history of acute knife-like pains in the epigastric region, collapse, subnormal temperature, rigid abdomen, pale grayish face, and by x-ray an airbubble under the diaphragm. In comparing black widow spider bite, besides the history of the bite, the pain spreads from the area of the bite to the abdomen. Also there are symptoms of generalized muscular pain and spasm, emotional instability, hypersensitivity of reflexes. Quick response to the treatment for latrodectus mactans bite will confirm the syndrome of arachnidism. Laboratory tests are of very little value in these cases. There is usually a slight leucocytosis and slightly increased polynuclear count, but there is no noted increase of eosinophils. Urine is usually negative. Only in extremely severe cases are found albuminuria, casts, and an increase of RBC. Some authors tells of increased blood urea in terminal cases.

Advocated treatments for black widow spider bites have been many. First and most important is the use of specific antivenom. Antivenom should be used as early as possible after the bite: delayed treatment does not give good results. We were giving a single dose of 2.5cc of "lyovac" antivenom (latrodectus mactans), a Sharp & Dohme product. In our first case antivenom was given one-half hour after the bite, two hours, six hours, and nine hours. In the first case comparatively mild symptoms developed. About 16 hours after the bite the patient had no specific complaints. In the second case, pains subsided within one hour after antivenom was given. In the two other cases symptoms subsided very slowly, and active additional therapy was required.

10cc of 20% sol. of magnesium sulfate or 10cc of 10% sol. calcium gluconate by intravenous injections gives prompt but temporary relief of pain. It is best to alernate both drugs, which we did in one case with good results.

The use of morphine and other opiates in combination with atzopine does not give the expected good results, but is valuable to fight restlessness. Bell and Boone and Beck are reporting good results by the use of 2cc of 1:2000 neostigmine methylsulfate alone or in combination with atropine sulfate gr. 1/150 to counteract the muscarinic effects of neostigmin.

When there is neck rigidity and severe headaches, lumbar puncture has been suggested as a method of affording some relief. In certain cases hot baths give some additional relief.

Local treatment of incision and suction has been tried by some authors, but this procedure is generally of no value, as the toxin is very rapidly absorbed. Hartinger reported good results using locally gauze saturated with 5% solution of gold chloride. Some mention using iodine and ichthyol locally.

Prognosis is favorable, considering the high toxicity of the venom, but there are reports giving a mortality rate of two to three percent. In our five cases there was one death, but here the diagnosis was questionable. Factors affecting the prognosis are age, health, and location of the bite. Alcoholics and syphilitics appear to experience more serious consequences than healthy human beings.

Immunity, according to D'Amour, is acquired slowly, after repeated injections of the venom. Several bites in one individual, with diminishing reaction of arachnidism, is recorded.

Protective measures include fine mesh screenings to keep out insects. Use mosquito netting when sleeping outdoors. Inspection of sleeping bag and clothes may be considered as a valuable prophylactic measure. Spraying with crue oil alone, or with a mixture of creosote will destroy the spider. Powdered unslacked lime and kerosene will kill the adult spider in about thirty minutes. All webs, spiderlings, and eggs should be destroyed by careful spraying or burning. It is very important to thoroughly spray outdoor privies and to inspect them before using again. Outdoor privies are considered one of the most common places of black widow spider infestation. The most efficient natural enemy is reported to be the mud dauber wasp.

The ability of the physician to recognize and to quickly relieve the symptoms of arachnidism is especially life-saving to very young children and the aged.

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Dr. Springer Honored

Dr. Harold L. Springer of Centerville was honored for his 50th year in medical practice by his friends and colleagues at a testimonial dinner recently at the Du Pont Country Club.

Dr. Springer was presented with a gold desk clock, given on behalf of those present by Dr. Sylvester W. Renie. Dr. G. W. K. Forrest was toastmaster.

Talks in tribute to Dr. Springer were given by Dr. George Wilson and Dr. Thomas Kline of Philadelphia. Dr. Springer told some of his experiences in medicine.

About 75 Delaware physicians attended, including his staff associates at the Delaware Hospital. Dr. Springer is a consultant in the surgery division and a former chief surgeon.

Among those at the dinner were Dr. Springer's brother, Willard Springer, Jr., president of the Industrial Trust Company, and his two sons, Harold L. Springer, Jr., and William L. Springer. William C. Northrop, a member of the board of the hospital, also attended.

Dr. Springer was graduated from the Wilmington High School in 1898, and received his degree of M. D. from the University of Pennsylvania in 1902. After a two-year interneship at Presbyterian Hospital, Philadelphia, he established a practice in Wilmington in 1904.

HORSE SENSE

Something possessed by horses that keeps them from betting on people.

BOOK REVIEW

Clinical and Roentgenologic Evaluation of the Pelvis in Obstetrics. By Howard C. Moloy, M. D., Assistant Clinical Professor of Obstetrics and Gynecology, Columbia University. (American Monograph Series) Pp. 119, with 68 figures. Paper. Price, $2.50. Philadelphia: W. B. Saunders Company, 1951.

One of the important problems of obstetrics today is difficult labor. Because disproportion is the chief cause of difficult labor, many attempts have been made in the past to devise methods to detect this condition.

In 1933, following much clinical and x-ray study of the female pelvis, Caldwell and Moloy devised a classification based upon morphological lines and included four parent types and numerous mixed types.

In this monograph the author has described the variations in the female pelvis which are of obstetrical significance and has correlated these with their effect on labor. He has shown how, by clinical and roentgenologic methods of examination, the various abnormalities may be recognized and dealt with.

This monograph is recommended to the medical student and every physician who does obstetrics. It can well be read with profit.

A Simple Definition

The "Gulf Breeze" house organ of the Gulf Insurance Company of Dallas cites the following definition as found in Britain's Health Act: "The expression 'infectious disease' means primarily any disease included in regulations under Subsection 1 of Section 29, whether absolutely or by definition of a particular stage of such disease, but in any section of Part IV of this act from the application from which a disease or a stage of disease is excluded under Subsection 2 of said Section 29, the expression does not include such disease or such disease in such stage as the case may be." Everything clear now? (*The Insurance Record*).

Three out of four traffic accidents happen in clear weather on dry roads.

Male drivers in 1951 were involved in 90 per cent of all U. S. auto accidents.

Saturday is the most dangerous day of the week in traffic.

The hour from six to seven p.m. is the most dangerous of the day or night in traffic.

+ Editorials +

DELAWARE STATE MEDICAL JOURNAL

Owned and published by the Medical Society of Delaware, a scientific society, non-profit corporation. Issued about the twentieth of each month under the supervision of the Committee on Publication.

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Articles are accepted for publication on condition that they are contributed solely to this JOURNAL. Manuscripts must be typewritten, double spaced, with wide margins, and the original copy submitted. Photographs and drawing for illustrations must be carefully marked and show clearly what is intended.

Footnotes and bibliographies should conform to the style of the Quarterly Cumulative Index Medicus, published by the American Medical Association, Chicago.

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Subscription price: $4.00 per annum, in advance. Single copies, 50 cents. Foreign countries: $5.00 per annum.

VOL. 24 JUNE, 1952 NO. 6

THE NEW ROSTER

With the May issue of THE JOURNAL there was sent to every one of the 335 members of the Medical Society of Delaware a copy of the New Roster. As stated thereon, we are quite certain it contains some (we hope only a few) errors. Prompt corrections are sought *in writing*, together with additions and changes as they occur. On the back page is provided space for additions for the next two years, which will be published in THE JOURNAL from time to time. Keep your copy of the Roster up to date: use a typewriter, as the calendered paper does not take ordinary ink very well.

On account of the high cost and the comparatively few additions and deletions each year, the Roster is published biennially. Even then it requires four months of exacting work, but it winds up the most complete and informative Roster that we have yet seen issued by any state medical society.

It takes only a momentary glance to realize that our membership contains a very high percentage of specialists, meaning 169 are full-time and 23 more are part-time specialists; or 50.4 and 6.8 percent respectively, making a total of 57.2 percent, and leaving only 42.8 percent of our members to work as general practitioners. Serious students of our American medical services have been saying for years that our profession is overspecialized: this new Roster seems to confirm that criticism. These same students indicate that the optimum is about one-third specialists and two-thirds general practitioners.

THE W. A. DONATES

The Delaware campaign for contributions to the American Medical Education Foundation, under the direction of Dr. C. L. Hudiburg, is beginning to show results. The individual doctor is being asked to make any donation to his own alma mater via this new A.M.E.F., in order to encourage contributions from non-medical sources. For full details see the editorial in THE JOURNAL for February, 1952, page 55.

We are now informed that the Woman's Auxiliary of the Medical Society of Delaware and of the New Castle County Medical Society have each contributed $100 to this Foundation. We hasten to say that these generous gifts evoked for these Auxiliaries our admiration and our sincere thanks: they are very much appreciated. Last year the National Auxiliary gave $10,000 to the A.M.E.F., and set the pattern for the State and County Auxiliaries. Good work!

VOTING RECORDS OF CANDIDATES

Senator Robert A. Taft, (R), Ohio—One of medicine's stauchest friends in the United

States Senate. He is campaigning vigorously against Socialized Medicine and all forms of State Socialism. He voted *against* Reorganization Plan #1 which, if not defeated, would have created a Cabinet position for Federal Security Administrator Oscar Ewing.

General Dwight D. Eisenhower, (R)—He has made no public statement as yet on the issue of Compulsory Health Insurance or Socialized Medicine. He has spoken out against some socialistic proposals, but his leading backers in the Presidential race include several so-called "Fair Deal Republicans" whose position on the medical issue is at least questionable. General Eisenhower's position may be clarified within the next few weeks. In this regard, yesterday Senator Dirksen (R), Illinois, cabled Senator Lodge (R), Massachusetts, who is at Eisenhower's headquarters, asking that he get Eisenhower to make a public statement on eight key National issues. On our issue, Senator Dirksen specifically asked: "Is he (Eisenhower) for or against Socialized Medicine?"

General Douglas A. MacArthur, (R)—Outspoken opponent of Socialized Medicine, Government controls and all socialistic proposals.

Harold E. Stassen, (R)—Mr. Stassen took a strong position against Socialized Medicine in a series of articles written for The Reader's Digest in January and February of 1950, following a trip to England and a study of the British system.

Governor Earl Warren of California, (R)—A constant and determined advocate of Compulsory Health Insurance, even though he denies that this is Socialized Medicine. Governor Warren caused Compulsory Health Insurance legislation to be introduced at the 1945, 1947 and 1949 sessions of the California State legislature and fought vigorously, but unsuccessfully for its enactment. He has become a bitter critic of the medical profession and, if elected President, undoubtedly would sponsor National Compulsory Health Insurance legislation similar to that advocated by the Truman Administration. He has also said in recent addresses that he favors virtually all of the New Deal legislation enacted during the past 20 years, but feels he could administer the program better than the Democrats.

Senator Estes Kefauver, (D), Tennessee—In a letter to Dr. R. B. Robins of Camden, Arkansas, a member of the AMA Coordinating Committee, dated January 30, 1952, Senator Kefauver said: "As you know I have heretofore taken my position against the medical bill that is now in Congress. I don't want anything to happen that may bring about Socialized Medicine." Senator Kefauver, however, voted *for* Reorganization Plan #1, which would have made Mr. Ewing a Cabinet member, and has supported some other aspects of the New Deal program.

Governor Adlai E. Stevenson, (D), Illinois—The current issue of Newsweek (April 14, 1952) carries an interview with Governor Stevenson, dealing with major National issues. He was asked the question: "Do you favor compulsory national health insurance?" His answer, according to Newsweek, was: "Basically, the problem is how to lift people over the costs of major illness. I don't know whether voluntary plans can do the job. I think the new commission on medical needs may well add some light and remove some heat, enabling us to find a satisfactory solution to this perplexing problem."

Senator Robert S. Kerr, (D), Oklahoma—He voted *for* Reorganization Plan #1, which would have given Oscar Ewing Cabinet status, and has generally favored New Deal socialistic legislation.

Senator Richard B. Russell, (D), Georgia—An outspoken opponent of most socialistic legislation; he voted against Reorganization Plan #1.

Senator Harry F. Byrd, (D), Virginia—A vigorous opponent of Socialized Medicine and all forms of Socialism. He addressed the Los Angeles Mid-Winter meeting of the AMA on this issue, December 5, 1951.

Vice President Alben Barkley, (D)—He has supported most of the Fair Deal program. To the best of our knowledge, he has not taken a public position on Socialized Medicine.

By Whitaker & Baxter

In infant formulas...

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caloric

distribution

calls for

adequate added carbohydrate

FREQUENT mention in authoritative pediatric literature supports the classic caloric distribution of 15% protein, 35% fat and 50% carbohydrate for infant formulas.

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